

Case Report

Female, 64 yo, Caucasian 5 weeks of immobilization after malignancy abdominal tumor resection. Sudden-onset dyspnea, tachypnea, chest pain of a "pleuritic" nature (worsened by breathing), cough and hemoptysis.

Physical examination: a pleural rub audible over the right lung. Signs of acrocyanosis (lips and fingers). Signals suggestive of raised jugular venous pressure. An A-wave is detected on examination of the jugular venous pulse in the neck.

A right ventricular lifting impulse along the lower left sternal border, a palpable pulmonic valve closure, accentuated pulmonic component of the second heart sound.

An holosystolic murmur of tricuspid regurgitation is heard along the lower left sternal border. The murmur increase coincident with inspiration. BP 95/60mmHg.

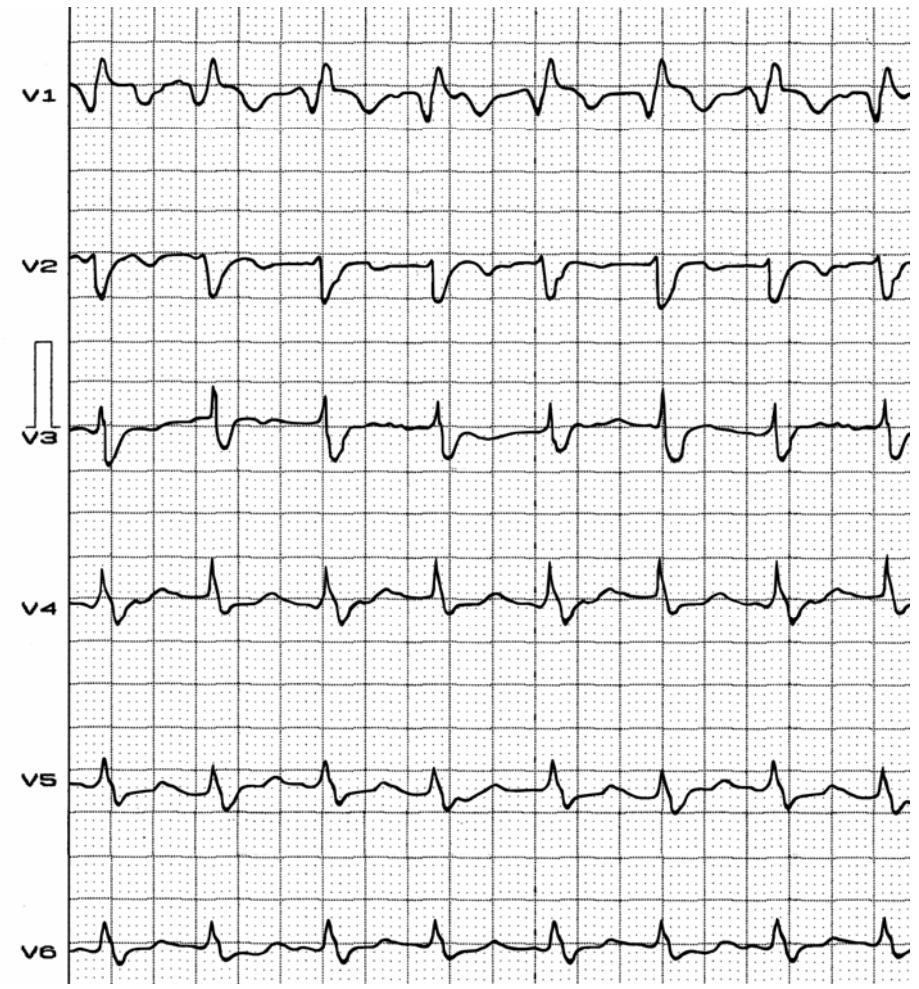
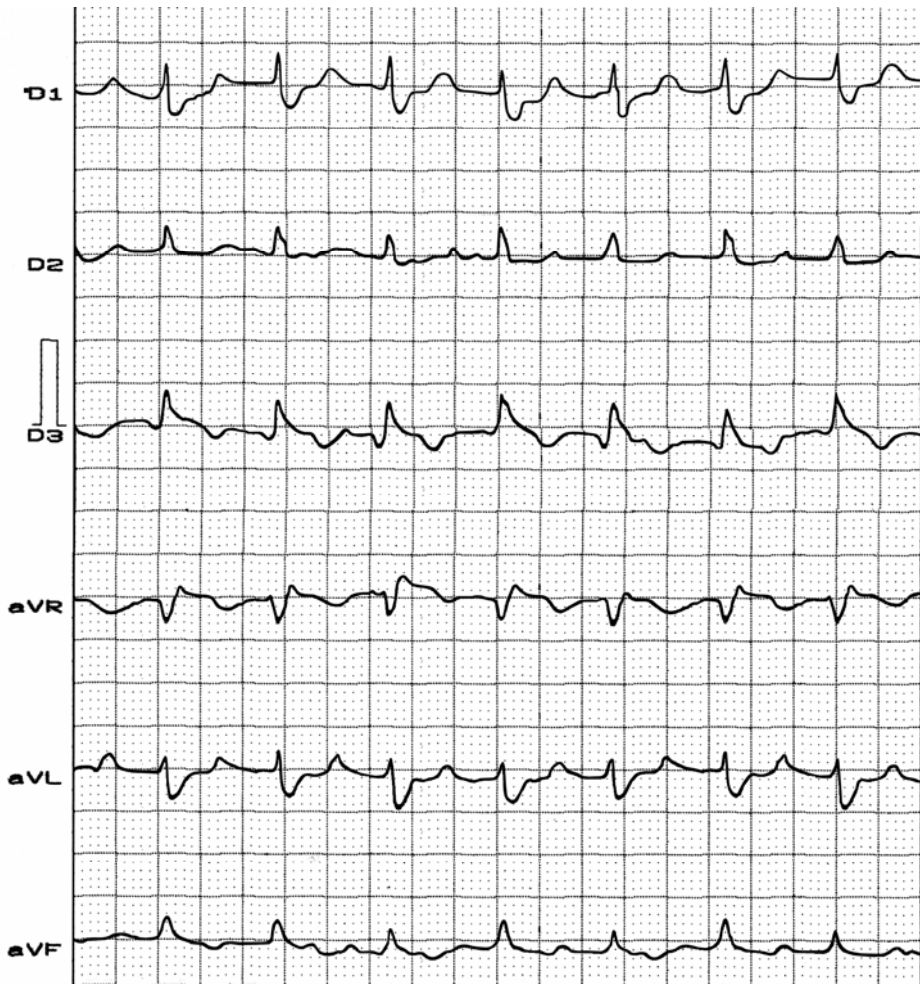
Femenina, blanca 64 años, inmovilizada 5 semanas después de la resección abdominal de tumor maligno.

Disnea repentina, taquipnea, dolor precordial de carácter pleurítico (peora al respirar), tos y hemoptisis.

Examen físico: acrocianosis de labios y dedos. Señales sugestivos de la presión venosa yugular elevada en la visualización del pulso venoso.

Se palpa un impulso de ventrículo derecho en el borde esternal izquierdo bajo, frote pleural sobre el pulmón derecho y el cierre del componente válvular pulmonar del segundo ruido.

Soplo holosistólico de regurgitación tricúspide se oye en el borde esternal izquierdo bajo. La intensidad del soplo aumenta con la inspiración. El componente pulmonar del segundo ruido se encuentra aumentado en intensidad. PA 95/60mmHg.



Dear Prof. Andrés Pérez Riera

It could be a case of Acute Pulmonary Embolism (APE): S1Q3T3 sign, qR sign in V1, RBBB, "silent" ST segment elevation in aVR, V1

QR in V1 sign is associated with a poor prognosis.

All the best from Poland

Peter Kukla, MD, PHD

PS. I send our last analysis concerning APE and ECG: **Combination of Electrocardiographic signs in differentiation between 2 syndromes with precordial negative T waves: acute coronary syndrome without ST elevation and acute pulmonary embolism (APE).**

Conclusion: The more promising ECG configuration helpful in different diagnosis between ACS and APE with negative precordial T waves are: NTW III + dextrogyria and \uparrow ST aVR + S1Q3T3 sign.

Querido Prof, Andrés Pérez Riera

Este puede se un caso de Embolia Pulmonar Aguda (EPA) patrón S1Q3T3, patrón qR en V1, BRD, "silente" elevación del ST en aVR, V1

QR en V1 se asocia a pronóstico ominoso.

All the best from Poland

Peter Kukla, MD, PHD

PS: Le estoy enviando nuestro ultimo trabajo sobre EPA y ECG titulado Combinación de signos en la diferenciación entre 2 sindromes con ondasT negativas: SCA sin elevación del ST e EPA

Conclusión: La configuración ECG que mas ayuda al diagnóstico diferencial entre SCA y EPA son onda T negativa en III, rotación horária en el eje longitudinal, supradesnivel del segmento ST en aVR y el patrón S1Q3T3.

Saludos desde Polonia

Peter Kukla, MD, PHD

Dear Andrés: For a more accurate judgment of the rhythm, an ECG prior to or post event should be provided (the way we teach our students). Otherwise with very limited info we are forced to make wild guesses like following which could be awfully off the track: 1. Atrial flutter?; 2. AV disassociation? (i.e. complete AVB); 3. Severe degree of IVCD (with a pattern similar to LPFB+CRBBB). With such poor ECG morphology and the acute symptoms described, the EP is likely <25% Thank you very much for sharing, and I look forward to hearing more from you.

Li Zhang, MD

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Estimado Andrés, Para un juicio más exacto del ritmo, un ECG antes del evento debería ser mostrado (la manera enseñamos a nuestros estudiantes). Si no, con el Info muy limitado nos fuerza a hacer conjeturas salvajes que puede hacernos salir terriblemente de la pista. 1. Aleteo atrial? ; 2. Disociación AV? (Ejemplo bloqueo AV completo); 3. Severo grado de estrés de las cámaras derechas (con un patrón que recuerda BDPI + BCRD LPFB). Con tal morfología pobre de ECG y los síntomas agudos descritos, el EP es probable Gracias por compartir y espero escuchar mas de ti.

Li Zhang, MD

Reply:

Dear Li your observation about LPFB is very interesting and clever, but Mauricio Rosenbaum teach us that the diagnosis of LPFB is possible only in absence of RVH. Interestingly 39 years ago Scott¹ wrote a manuscript questioning this point. What do you think Marcelo Elizari about it?

Thank in advance

Andrés Pérez Riera

Querida Li su observación sobre el bloqueo divisional póstero-inferior (BDPI) es muy interesante e inteligente, pero Mauricio Rosenbaum nos enseñó que el diagnóstico de BDPI solo es posible en ausencia de SVD. Interesantemente hace 39 años Scott¹ escribió un manuscrito interrogando este punto. Que piensas querido Marcelo Elizari acerca de esto?

Dear Andrés,

In medicine we all follow what the guideline says and the grandpa gold standards. But so few remind us the guidelines are not always perfect that sometimes we may have to challenge the old think by good reasoning and research.

Yes LPFB should be ruled out if patients with RVH could never develop such fascicular block. ECG wave patterns might not be able to separate LPFB and RVH from each other which does not necessarily mean RVH and LFB cannot coexist (though such combination is rare). In this case acute pulmonary embolism (APE, if it were true) may be causative to the dominant R waves in aVR and V1 if they were absent previously.

I apologize for overlooking the S1Q3T3 sign and greatly appreciate Dr. Peter Kukla for his sharp eye catch.

Thanks again for the great brain teaser testing,

Li

1. Scott RC. The S1Q3 (McGinn-White) pattern in acute cor pulmonale: a form of transient left posterior hemiblock? Am Heart J. 1971 Jul; 82:135-137.

Dearest Prof. Pérez Riera

Greetings

The clinical picture is highly suggestive of acute pulmonary embolism, as well the ECG: Tachycardia without clear P waves, some atrial activity is noticed but inconsistent. Right axis deviation, $S_1-Q_{III}-T_{III}$ sign which is highly specific but with low sensitivity to PE. This is a case of submassive PE. The "evidence based treatment" is: Full heparinization followed by long term warfarin. In case of more deterioration, then thrombolytic therapy may be indicated.

Raed Abu Sham'a, M.D. Palestine/Jerusalem

El cuadro clínico es altamente sugestivo de Tromboembolismo Pulmonar Agudo (TEPA), así como el ECG: Taquicardia sin ondas P claras. Se observa alguna actividad atrial pero esta es inconsistente. Desviación del eje a la derecha y el patrón $S_1-Q_{III}-T_{III}$ el cual es muy específico pero de sensibilidad baja para tromboembolismo pulmonar agudo.

Este es un caso de embolia pulmonar submasiva. Según los datos basados en evidencias debemos heparinizar en dosis plenas seguido de warfarina a largo plazo. En caso de mayor deterioro, la terapia trombolítica puede estar indicada.

Raed Abu Sham'a, M.D. Palestina/Jerusalem

Sin dudas que la clinica de la paciente, sumada a los antecedentes inmediatos quirúrgicos y de inmovilizacion de los que viene precedida, orientan a la sospecha de una TEPA. No me quedó claro si el BCRD es agudo o si ya existía dicho trastorno de conducción.

Respecto del tipico patron ECG de McWhite: SI QIII TIII, alguna literatura le asigna mayor importancia, mas en la actualidad, a los cambios en la repolarizacion en las precordiales derechas. Este último comentario, seguramente podra ser aclarado, por los calificados miembros (notables) que integran este foro.

Dr. Francisco E. Viano Argentina

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Without doubts that the patient clinic of, added to the surgical immediate antecedents and immobilization from which it comes preceded, orient to the suspicion of APE.

I'm not sure if the RBBB is acute or if already exist before the event.

With respect to typical Mc Ginn White ECG pattern(S1-Q3-T3) some literature assigns to major importance at present to the changes in repolarizacion in the right precordial leads. This last commentary, surely will be clarify, by the remarkable members integrant of this forum.

### **Dr. Francisco E. Viano Argentina.**

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**A/V Dissociation with junctional rhythm, RBBB with qR pattern in V<sub>1</sub>. S in lead I and Q in lead III with late R wave transition. ECG shows evidence of severe pulmonary hypertension. The qR in V<sub>1</sub> in my experience should be more reflective of chronic hypertension. She may have had repeated episodes.**

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**Disociación AV con ritmo juncional, BRD, con patrón Qr en V1. Este patrón qR en mi experiencia debería hacernos pensar e hipertensión pulmonar crónica. S<sub>I</sub>-Q<sub>III</sub>. Zona de transición tardía.**

**El ECG muestra evidencias de severa hipertensión pulmonar. La paciente puede haber tenido repetidos episodios.**

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**Prof. Melvin Scheinman M.D. Ph.D.**

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**Se me ocurre que es un corazón pulmonar agudo, por la sintomatología. Pero a la vez hay una pericarditis por la proximidad de la pleura que da el roce y el dolor precordial. Ingurgitación yugular, mala oxigenación(acrocianosis), aumento de la presión pulmonar(cierre del componente pulmonar aumentado en intensidad), soplo holosistólico de regurgitación tricuspídea (insuficiencia por aumento de las cavidades derechas). Disnea, taquipnea, proceso pulmonar. ECG: Taquicardia sinusal (130 x min), bloqueo AV 1er grado (PR= 280 mseg). bloqueo completo de rama derecha. -----**

**I think that is an APE by the symptomatology. But while there is pericarditis by the proximity of the pleura which gives the friction and chest pain. Jugular venous distention, poor oxygenation (acrocyanosis), increased pulmonary pressure (closure of the pulmonary component increased in intensity), holosystolic murmur of tricuspid regurgitation (shortfall by increasing the right cavities). Dyspnea, tachypnea, pulmonary process.**

**ECG: Sinus Tachycardia (130bpm). 1st degree A-V block (PR=280 msec) and CRBBB. -----**

**Eu acho que é um APE pela sintomatologia. Além disto pericardite pela proximidade da pleura que ocasiona o atrito e a dor torácica. Distensão venosa jugular, má oxigenação (acrocianose), aumento da pressão arterial pulmonar (aumento na intensidade do componente pulmonar da segunda bulha), sopro holossistólico de regurgitação tricúspide (déficit através do aumento das cavidades direitas). Dispnéia, taquipnéia, processo pulmonar.**

**ECG: Taquicardia sinusal (130x´), bloqueio AV de 1 grau (PR = ms 280) e BCRD.**

**Cordialmente/Sincerely/Atenciosamente**

**Dr. Eduardo Quiñones Argentina.**

**Me animo a opinar ante gente de tan elevado nivel con el objetivo de aprender.**

**¿Por que se produciría un ritmo de la unión en esta paciente?**

**¿Cual seria el mecanismo?. Además, la onda P tiene una frecuencia elevada. Si yo conté bien, en DIII parece tener una frecuencia de 150 latidos por minuto. Por otra parte, en esta misma derivacion aparecen ondas negativas lo que sugeriría activación baja de la aurícula.**

**No me parece flutter con sobrecarga aguda del ventrículo derecho. El hacer un diagnostico diferencial entre flutter y taquicardia de la unión a esta paciente que aportaria?**

**Y ya lo que me agradó como la siguieron y como terminó todo queria saber: ¿Tenia falla aguda del VD? ¿que hace pensar en esta posibilidad segun los datos del ECG ?**

**Dr. Simon Pero [simonpero@YAHOO.COM](mailto:simonpero@YAHOO.COM) Argentina.**

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**My intention to give my opinion in front of people of so high level with the aim to learn.**

**What produce AV junction rhythm in this patient?**

**Which is mechanism?. In addition, the P wave has high frequency. If I counted well, in DIII it seems to have a frequency of 150 bpm. On the other hand, in this same derivation appear negative waves that suggest low activation of the atrium. It does not seem to me a flutter with acute overload of the right ventricle. Doing differential diagnostic between flutter and Junction AV tachycardia to this patient who contribute to her?**

**I would like to know if the patient had acute failure of the right ventricle. What do to think about this possibility in the ECG analysis?**

**Dr. Simon Pero [simonpero@YAHOO.COM](mailto:simonpero@YAHOO.COM) Argentine.**

- a) Resulta difícil establecer el ritmo que comanda las aurículas.
- b) Hay numerosos elementos que apoyan el diagnóstico de TEPA:
  - 1) Rotación horaria franca ( $S_1$ -  $Q_{III}$ )
  - 2) Supradesnivel del segmento ST en aVR
  - 3) Onda Q grande en  $V_1$  indicando dilatación de aurícula derecha.

Hay retardo en la conducción por la rama derecha. Ante cuadro de TEPA esperaría -en caso de ritmo sinusal - P acuminadas especialmente en DII  $V_1$ . Al no tener estos detalles, pienso que puede existir una FA. Los ventrículos están activados por un ritmo de la union de frecuencia mayor a 100pm (aproximadamente 115pm) por lo que podemos hablar de taquicardia de la union.

Cordialmente

**Dr. Carlos Lavergne, NEUQUÉN: PATAGONIA ARGENTINA**

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- a) It is not easy to know which is the atrial command
- b) I observe several indicatives of Acute Pulmonary Embolism (APE):
  - 1) CCW rotation ( $S_1$ - $Q_{III}$ )
  - 2) ST segment elevation in aVR lead
  - 3) Big initial Q wave in  $V_1$  suggestive of right atrial dilatation

There is right end conduction delay by the right bundle. A clinical picture of APE in sinus rhythm would have acuminate P waves; maybe AF. Ventricles are activated by a tachycardial junction rhythm with HR>100bpm( approximately 115bpm).

Sincerely

**Dr. Carlos Lavergne from NEUQUÉN: ARGENTINE PATAGONIA (very cold!)**

# Differential Diagnosis of Acute Chest Pain

1. Acute Coronary Syndrome (ACS)
2. Acute Pulmonary Embolism (APE)
3. Aortic dissection
4. Aortic valve disease
5. Hypokinetic cardiomyopathy
6. Stress cardiomyopathy
7. Dilated cardiomyopathy
8. Myocarditis
9. Pericarditis
10. Lung parenchyma abnormalities: Pneumonia, carcinoma, pneumotórax.
11. Hiatus hernia, esophagitis, ulcer, esophageal carcinoma
12. Sepsis
13. Trauma
14. Arrhythmias
15. Tietze syndrome
16. Herpes zoster
17. Intercostal neuralgia
18. Metastatic carcinoma
19. Osteomyelitis
20. Diaphragmatic abscess.

1. Bierry G, Roy C, Buy X, Kellner F, Jlassi H, Gangi A. ECG-gated chest CT angiography: value for atypical chest pain evaluation *J Radiol.* 2009 Jul-Aug;90:825-831.
2. Nunes JP. Pseudo myocardial infarction - A condition in need to be redefined? *Med Hypotheses.* 2009 Oct 23. [Epub ahead of print]

# Our ECG diagnosis

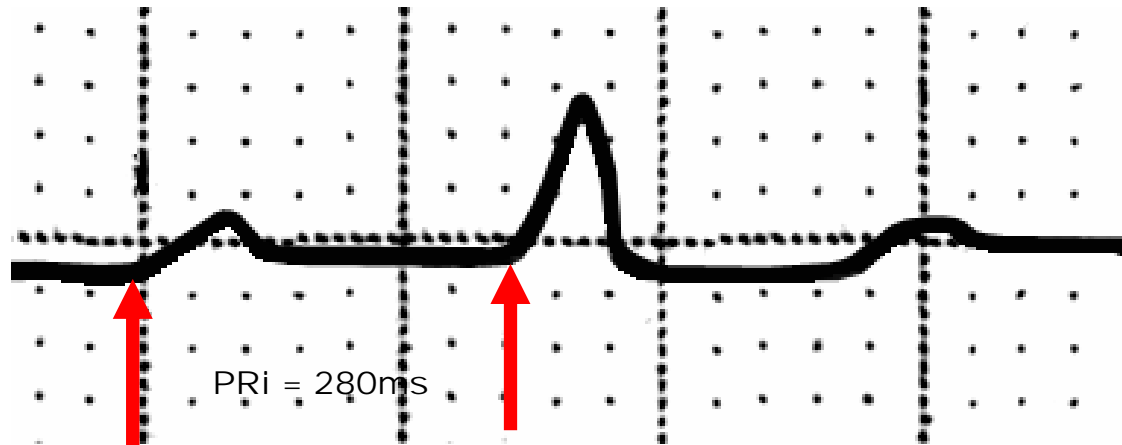
1. Rhythm and Heart rate: Intermittent sinus tachycardia: 108bpm. Eventual possible AV dissociation with Tachycardial Junctional comand. When sinus comand is present prolonged PR interval : first-degree AV block( neuropraxis) see next slide
2. PR interval: first-degree AV block( neuropraxis) see next slide
3. QRS axis: Right axis deviation  $\approx +120^\circ$
4. QRS duration: 115ms
5. QRS complex: QRS duration:115ms, Clockwise Rotation (**CW**) with shift of the transitional zone to the left, low r waves and relatively deep S waves across all precordial leads, incomplete Right Bundle Branch Block pattern(IRBBB): broad final S waves in I, aVL, V<sub>5</sub>-V<sub>6</sub>, qr pattern in V1: Sodi-Pallares sign<sup>1</sup>: qR, qr, QR or qRs in V1 and V2. QRS with initial q in V1 or V1 and V<sub>2</sub> indicative of significant right atrial dilatation, diffuse low QRS voltage in both frontal plane (FP) and horizontal plane (HP), signs of right heart strain: Large S wave in lead I, large Q wave in lead III and "S<sub>I</sub>Q<sub>III</sub>T<sub>III</sub>" pattern.
6. ST segment: minimal ST segment elevation in aVR, III and V<sub>1</sub>.
7. T wave: T wave inversion in lead III

6 and 7 are findings that could mimic Acute Inferior Myocardial Infarction.

In a prospective cohort study of unselected patients with suspected APE although there are ECG changes that are more common in APE, the ECG alone is not sufficiently sensitive or specific to rule out or rule in the diagnosis<sup>2</sup>. ECG changes can be neither sensitive nor specific for the diagnosis of massive APE<sup>3</sup>.

1. Sodi-Pallares D, Bisteni A, Herrmann GR. Some views on the significance of qR and QR type complexes in right precordial leads in the absence of myocardial infarction. *Am Heart J.* 1952 May; 43: 716-734.
2. Brown G, Hogg K. Best evidence topic report. Diagnostic utility of electrocardiogram for diagnosing pulmonary embolism. *Emerg Med J.* 2005 Oct; 22: 729-730.
3. Prasad UK, Berkin KE. How ECG can cause confusion in pulmonary embolism and how echocardiogram can help. *Emerg Med J.* 2006 Jan;23(1):e2.

# Prolonged PR interval



Tachycardia with intermittent A/V dissociation with Tachycardial Junction Rhythm, but clearly intermittent significant PR interval prolongation consequence of NEUROPRAXIA phenomena.

When the right atrium is enlarged, transmission of the impulse from the SA to the AV node is slightly delayed because neuropraxia on AV node occur\* as consequence of direct pressure on right AV region. (such a Ebstein's anomaly). With the availability of echocardiography, the ECG terms of enlargement was changed to right and left atrial abnormalities, because of the relative inadequacy of ECG in predicting atrial size.

In this case, Macruz index<sup>1</sup>, an indirect sign of right atrial enlargement is < 1. **Macruz**

$$\text{Index} = \frac{\text{P duration}}{\text{PR segment}}$$

\*Condition in which conduction is blocked at a point in the nerve or conduction system but is present above and below that point.

# MACRUZ INDEX

$P/PRs$  = **The normal value of the Macruz index is 1 to 1.8.**

**This index may be useful in diagnosis of atrial overload. Thus, in left atrial enlargement (LAE) by increase in the numerator's duration (P wave) the P/PRs index turns out to be greater than 1.8.**

**In right atrial enlargement (RAE), an increase in the denominator's duration (PRs segment) is observed, with normal P duration, which causes a P/PRs ratio with values under 1.**

**In the presence of biatrial enlargement (BAE), an increase is observed both in P and PRs duration. Thus, the index is normal with increased PRs.**

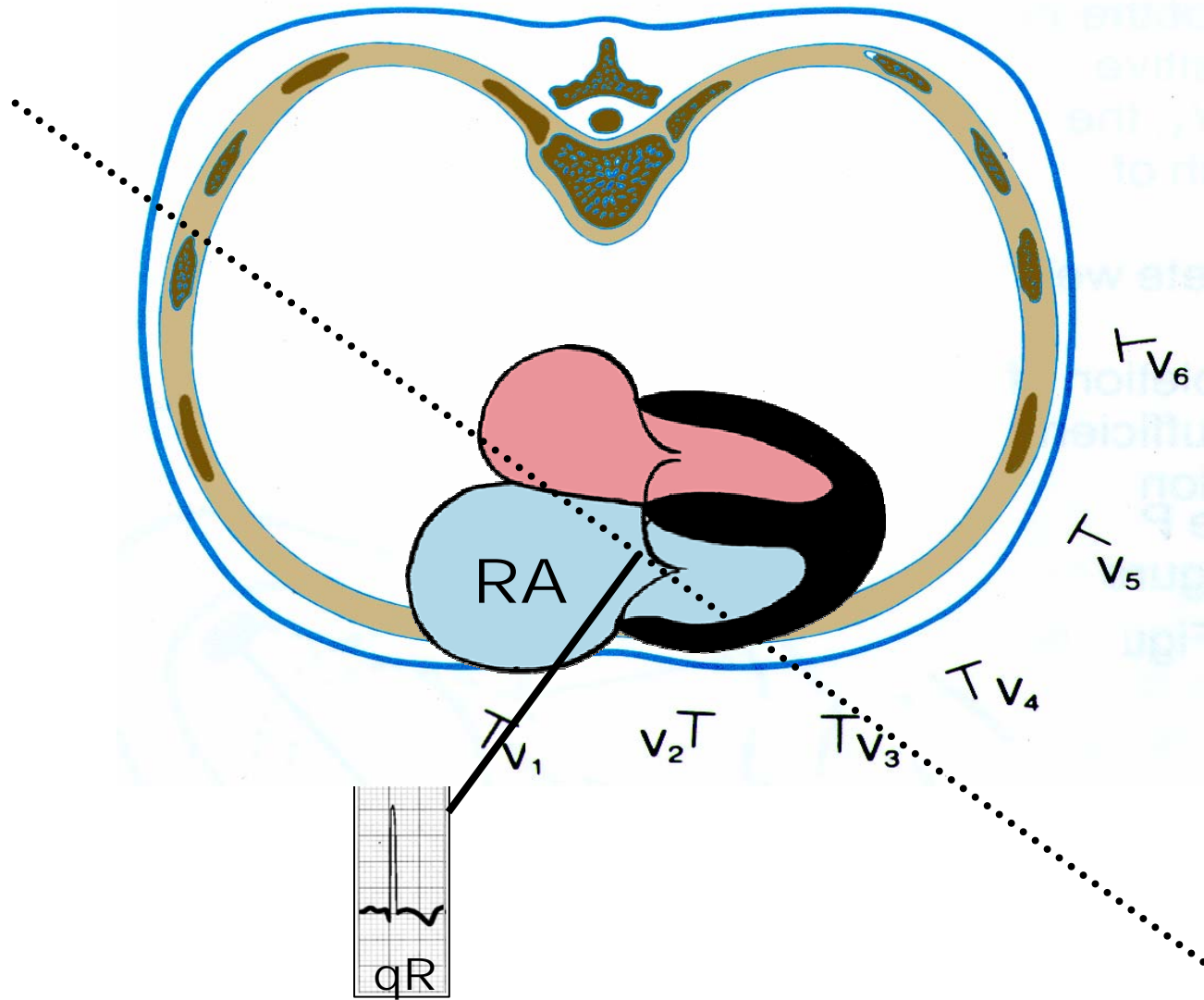
# The possible causes of qR pattern in right precordial leads

1. Severe Systolic Right Ventricular Overload “extreme strain pattern”: suprasystemic right intraventricular pressure. E.g.: Severe Pulmonary Stenosis<sup>1</sup>.
2. Extreme dilatation of the Right Atrium (RA), aneurismal RA, or Right Atrial Enlargement (RAE): qR pattern in V<sub>1</sub> may be an indirect sign of RAE E.g.: Ebstein's anomaly<sup>2</sup>, tricuspid insufficiency. Characteristically, Ebstein's anomaly shows in V<sub>1</sub> lead bizarre and low voltage RBBB with initial q wave. In Ebstein's anomaly, the absence of RBBB pattern is a strong predictor of an accessory A-V pathway<sup>3</sup>. Electrocardiographically, the combination of the “Himalayan P waves” and the low voltage atypical RBBB with initial q wave and without right ventricular overload almost certainly constitutes a pathognomonic finding in Ebstein's anomaly. The height of the P wave and the duration of the QRS complex are related to the severity of the anomaly. The volumetric increase of the RA gets it closer to the exploring electrode of V<sub>1</sub>, registering negatively initially in this lead, because the electrode records the epicardial morphology of the RA<sup>4</sup>.

1. Gandhi MJ, Datey KK, Kulkarni TP, Hansoti RC. Genesis of qR pattern in right precordial leads in right ventricular overload. J Assoc Physicians India. 1962 May;10:217-223.
2. Kumar AE, Fyler DC, Miettinen OS, Nadas AS. Ebstein's anomaly. Clinical profile and natural history. Am J Cardiol. 1971; 28: 84-95.
3. Iturralde P, Nava S, Sálica G, Medeiros A, Márquez MF, Colin L, Victoria D, De Micheli A, Gonzalez MD Electrocardiographic characteristics of patients with Ebstein's anomaly before and after ablation of an accessory atrioventricular pathway. J Cardiovasc Electrophysiol. 2006 Dec;17:1332-1336.
4. Sodi-Pallares D, Bisteni A, Herrmann GR. Some views on the significance of qR and QR type complexes in right precordial leads in the absence of myocardial infarction. Am Heart J. 1952 May;43:716-734.



# SODI-PALLARES'S SIGN



The volumetric increase of the right atrium (RA), gets closer to the exploring electrode  $V_1$ , recording initial negativity in this lead, because this electrode records the epicardial morphology of the RA.



**From right to left João Tranchesi, Prof. Dr. Demétrio Sodi-Pallares and Dr. Dante Peñaloza  
From the famous Instituto Nacional de Cardiología Ignacio Chávez, México.**

3. Congenitally Corrected Transposition: Secondary to inversion of septal activation, RAE, by progressive tricuspid regurgitation that occurs with age and associated with deterioration of RV function<sup>1; 2</sup>.

4. Chronic Obstructive Pulmonary Disease(COPD) : the presence of a qR pattern with delayed R wave in V1 (V5R) (onset of intrinsicoid deflection >30ms) which was often associated with an incomplete RBBB, “P pulmonale” and inversion of the T waves in standard and precordial leads were considered as labile, reversible ECG changes which do not show any correlation with the severity of COLD. Deviations of the P wave and QRS complex to the right, significantly correlated with the degree of the air-flow obstruction (P less than 0,05) in patients with COLD, but the changes are not sure signs of chronic cor pulmonale. On the contrary, qR pattern with delayed R wave in V1 (V5R) which was often associated with an incomplete RBBB, was significantly correlated with the degree of air-flow obstruction in patients with COLD and may be considered as highly suggestive for the diagnosis of chronic cor pulmonale<sup>3</sup>.

5. Acute Pulmonary Embolism

1. Warnes CA. Transposition of the great arteries. *Circulation* 2006; 114: 2699-2709.
2. Ruttenberg HD, Elliott LP, Anderson RC, Adams P Jr, Tuna N. Congenital corrected transposition of the great vessels. Correlation of electrocardiograms and vector cardiograms with associated cardiac malformations and hemodynamic states. *Am J Cardiol.* 1966; 17: 339
3. Kes P, Strumberger I, Reiner Z. Electrocardiographic changes in chronic obstructive lung diseases. *Z Gesamte Inn Med.* 1982 Dec;37:796-801.

**6. Complete RBBB associated to Anterior Myocardial Infarction:** analysis of HERO-2 trial demonstrates that in the setting of an anterior STEMI, the presence of an RBBB, whatever its onset, is associated with a higher risk of death<sup>1</sup>. In patients with anterior AMI and RBBB, increasing QRS duration is associated with increasing 30-day mortality. Early ST-segment resolution after fibrinolytic therapy despite persisting RBBB is associated with lower mortality rate<sup>2</sup>. Additionally, the presence of QS or QR complexes in both V4R and V3R are specific markers of right ventricular necrosis (specificity 100%; sensitivity 78%)<sup>3</sup>. RBBB accompanying anterior AMI at presentation and new BBB (including LBBB) early after fibrinolytic therapy are independent predictors of high 30-day mortality. These ECG features should be considered in risk stratification to identify high-risk patients<sup>4</sup>.

**7. Complete RBBB with isoelectric initial r wave in V<sub>1</sub>**

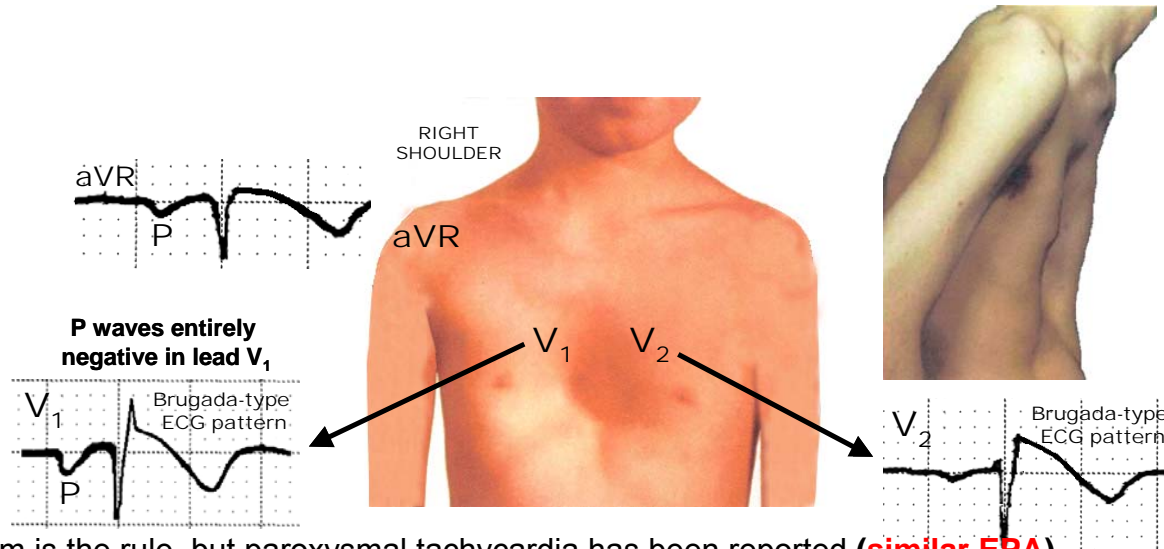
**8. Situs inversus:** ventricular inversion, Congenitally Corrected Transposition: Secondary to inversion of septal activation, RAE, by progressive tricuspid regurgitation that occurs with age and associated with deterioration of RV function<sup>5</sup>

**9. Pectus excavatum, funnel chest' or “sunken chest”**

**10. Endomyocardial fibrosis<sup>6</sup>.**

1. Wong CK, Gao W, Stewart RA, et al.; Hirulog Early Reperfusion Occlusion (HERO-2) Investigators. Risk stratification of patients with acute anterior myocardial infarction and right bundle-branch block: importance of QRS duration and early ST-segment resolution after fibrinolytic therapy. *Circulation*. 2006 Aug 22;114:783-739.
2. Di Chiara A, Marinigh R. Bundle-branch block in the course of myocardial infarction. *Recent advances Recenti Prog Med*. 2006;97: 401-404
3. Morgera T, Alberti E, Silvestri F, et al. Right precordial ST and QRS changes in the diagnosis of right ventricular infarction. *Am Heart J*. 1984 Jul;108:13-18.
4. Wong CK, Stewart RA, Gao W, et al. Prognostic differences between different types of bundle branch block during the early phase of acute myocardial infarction: insights from the Hirulog and Early Reperfusion or Occlusion (HERO)-2 trial. *Eur Heart J*. 2006 Jan;27:21-28.
5. Warnes CA. Transposition of the great arteries. *Circulation* 2006; 114: 2699-2709.
6. Tobias NM, Moffa PJ, Pastore CA, et al. The electrocardiogram in endomyocardial fibrosis *Arq Bras Cardiol*. 1992 Oct;59: 249-253.

# Pectus excavatum, 'funnel chest' or "sunken chest" and qR in right precordial leads ECG similarities between Pectus Excavatum and APE



**Rhythm:** sinus rhythm is the rule, but paroxysmal tachycardia has been reported (**similar EPA**).

**P waves:** entirely negative in lead V<sub>1</sub>, because the relation between the atria and the location of lead V<sub>1</sub> electrode is altered: This is explained by atrial activation proceeding in a direction away from the site of the lead V<sub>1</sub> because of leftward displacement of the heart owing to the chest deformity. Become similar to aVR frontal unipolar lead<sub>2</sub>. (**similar EPA**)

**QRS axis:** Occasionally extreme left QRS deviation (**similar EPA**).

**Rotation on longitudinal axis:** Clockwise rotation of the QRS complex in the precordial unipolar leads (**similar EPA**).

**qR pattern:** Abnormal q or Q waves are seen most often in the right and mid-unipolar precordial leads or in inferior leads (pseudo anteroseptal or inferior infarction)<sub>6</sub> (**similar EPA**).

**Triphasic pattern on right precordial leads:** is one of the characteristics ECG changes: PSEUDO INCOMPLETE RBBB. The last r' wave is usually small<sub>5</sub>.

**S<sub>I</sub>-S<sub>II</sub>-S<sub>III</sub> pattern** Sometimes is registred (**similar EPA**).

**T waves:** Non-ischemic-type T wave inversion in right or mild precordial leads (**similar EPA**)

**Brugada type 1 ECG patten:** Rarely observed (**similar EPA**)

1. De Oliveira JM, Sambhi MP, Zimmerman HA. The electrocardiogram in pectus excavatum. Br. Heart J 1958; 20:495-501
2. Dessler W, Roesler H. Electrocardiographic changes in funnel chest. Am Hear J 1950; 4: 877-883
3. Kataoka H. Electrocardiographic patterns of the Brugada syndrome in 2 young patients with pectus excavatum. J Electrocardiol 2002; 35:169-171.

# Monotonous ECG

Why monotonous ECG? Because the ECG aspect of the QRS complexes are repetitive. They are tediously repetitious or lacking in variety. Unvarying in pitch or cadence:

From the 12 leads we have 7 leads with the same pattern:

- rS or rs: I, aVL, V<sub>2</sub>, V<sub>3</sub>, V<sub>4</sub>, V<sub>5</sub> and V<sub>6</sub>
- aVR, III and V<sub>1</sub>: qr pattern and
  - II and aVF pure r wave.

## Low QRS Voltage

There are criteria of low QRS voltage of QRS complexes in both planes:

IN THE FRONTAL PLANE

- No wave exceeds **5 mm** (1 large square or 5 small squares, vertically) in the leads of the FP

IN THE HORIZONTAL PLANE

- No wave exceeds **10 mm** as the border in this plane.

# Transitional Zone on Precordial Leads

Definition: It is the site on precordial leads in which the amplitudes of the positive and negative deflections are of equal magnitude (isodyphasic QRS complex:  $R = S$ ). In normal adults it is usually located between lead  $V_2$  and  $V_4$ . The most common lead is  $V_3$ .

**Counter-Clockwise Rotation (CCW):** When transitional zone is located to right of  $V_1$  in adults.

**Clockwise Rotation (CW):** When transitional zone is dislocated to left located beyond lead  $V_5$  (as the present case).

About two-thirds of **CCW** and **CW** can be explained by anatomical rotation of the heart in one plane around the long axis (the septal angle), but other factors appear to be responsible for such electrocardiographic findings in the remaining one-third of cases as higher positions of the precordial ECG leads, as observed in the vertical heart and **Left Septal Fascicular Block<sup>1</sup>**.

1. Tahara Y, Mizuno H, Ono A, Ishikawa K. Evaluation of the electrocardiographic transitional zone by cardiac computed tomography. *J Electrocardiol.* 1991 Jul;24:239-45.

ECG changes tend to appear early and are characteristically transient. It is important serial tracing

**1. Classic Findings<sup>1</sup>:** SI QIII TIII (seen in under 20% of cases)

- Deep S wave in lead I
- Prominent Q wave in Lead III, and aVF but not in II
- T wave inversion in lead III: Mc Ginn White pattern S<sub>I</sub>-Q<sub>III</sub>-T<sub>III</sub>

**2. Common Findings**

- Sinus Tachycardia
- Right atrial abnormality (P pulmonale) is observed eventually. QRS axis may become vertical or shift rightward: Right axis QRS deviation. Leftward shift of frontal plane QRS axis as a frequent manifestation of APE<sup>2</sup>.
- Right sided strain pattern
- Clock wise rotation with shift of the transition zone to the left
- Eventually Si-SII-SIII pattern.
- Transient Incomplete or Complete Right Bundle Branch Block
- QR or QS pattern in V3R, V1 and V3 with negative T wave in these leads.
- Findings that mimic Myocardial Infarction
- ST segment changes<sup>3</sup>.
- T wave changes: negative T wave in III and/or over right and midprecordial leads.
- Transient Atrial Fibrillation (new onset) or others atrial, tachycardias, and atrial premature contractions<sup>4</sup>.

1. Rodger M, Makropoulos D, Turek M, Quevillon J, Raymond F, Rasuli P, Wells PS. Diagnostic value of the electrocardiogram in suspected pulmonary embolism. *Am J Cardiol.* 2000 Oct 1;86:807-809.
2. Lynch RE, Stein PD, Bruce TA. Leftward shift of frontal plane QRS axis as a frequent manifestation of acute pulmonary embolism. *Chest.* 1972 May;61:443-446.
3. Yeh KH, Chang HC. Massive pulmonary embolism with anterolateral ST-segment elevation: electrocardiogram limitations and the role of echocardiogram. *Am J Emerg Med.* 2008 Jun;26:632.e1-3.
4. Ullman E, Brady WJ, Perron AD, Chan T, Mattu A. Electrocardiographic manifestations of pulmonary embolism. *Am J Emerg Med.* 2001 Oct;19:514-519.



**3. Rare ECG manifestations:** elevation of ST segment

**4. Exceptional manifestation:** Immediately LBBB disappearance after the onset of Acute Pulmonary Embolism (APE), from surface ECG. Recovery from APE result in reappearance of LBBB pattern. Delayed conduction of the previously unaffected right bundle branch result in roughly equivalent onset of ventricular activation is the most likely reason<sup>1</sup>.

The following patterns are suggestive of APE:

1. Mc Ginn White Pattern<sup>2</sup>: SI-QIII-TIII, with T inversion in III and right or right to middle precordial leads. McGinn and White describe the changes to the ECG during APE. Scott postulated that this patten could be a form of left posterior fascicular block<sup>3</sup>.
2. Recent axis shift
3. S<sub>I</sub>-T<sub>III</sub> pattern with transient incomplete or complete RBBB
4. Clockwise(CW) rotation on long axis: transition zone on precordial leads dislocate to left.
5. "P pulmonale";
6. Acute transient atrial fibrillation<sup>3</sup> or atrial arrhythmias.

1. Athar SM, Chin BS, Flint EJ. Transient disappearance of left bundle branch block pattern: an unusual ECG presentation of acute pulmonary embolism. *Postgrad Med J.* 2002 Sep;78:555-558
2. McGinn S, White PD. Acute cor pulmonale resulting from pulmonary embolism : its clinical recognition. *JAMA* 1935;114:1473.
3. Scott RC. The S1Q3 (McGinn-White) pattern in acute cor pulmonale: a form of transient left posterior hemiblock? *Am Heart J.* 1971 Jul;82:135-137.
4. Koerner SK, Malovany RJ. Acute respiratory insufficiency and cor pulmonale: Pathophysiology, clinical features and management. *Am Heart J.* 1974 Jul;88:115-119.

# The causes and consequences of ECG/VCG modifications in Acute Pulmonary Embolism

1. Dilatation of the right atrium: **rare “P pulmonale” (P>2.5mm in inferior leads), right P axis deviation, qR pattern in V<sub>3</sub>R-V<sub>1</sub>**
2. Compression of AV node: **PR interval prolongation, Macruz index <1.**  
**Macruz Index = P duration / PR segment**
3. Local or focal intraventricular block secondary to marked localized dilatation of the Pulmonary Outflow Tract: **transient incomplete or complete periferical RBBB( divisional block on free wall right ventricle, that we denominate right end conduction delay) different from troncular RBBB**
4. Acute Coronary insufficiency secondary to a fall in cardiac output: **ST segment elevation or rarely depressed and inverted T wave in right precordial leads and III and aVF**
5. Cardiac clockwise rotation around the longitudinal axis: **right axis deviation, S<sub>I</sub>-Q<sub>III</sub>-T<sub>III</sub> Pattern of Mc Ginn and White, transition zone deviated to left, deep final S waves in I, Qr or QR pattern in V<sub>1</sub>.**

# Sensitivity of ECG for the diagnostic of APE

In massive embolism, the ECG is normal in 6% of cases. Submassive embolism, 23 % have a normal ECG. The traditional manifestations of APE: SI-1QIII-TIII, RBBB, P pulmonale, or right axis deviation occur in only 26 % of cases. The most common ECG features are nonspecific T wave changes ( 42% ) and nonspecific abnormalities (elevation or depression) of the ST segment (41%). Left axis deviation occur in 7% an is as frequent as right axis deviation. Low voltage QRS complexes occurred in 6%. Atrial fibrillation or atrial flutter, appears to occur more typically in patients with APE who have preexistent cardiac disease. All of the varieties of ECG abnormalities disappeared in some of the patients by 2 weeks. Inversion of the T wave is the most persistent abnormality<sup>4</sup>.

1. Stein PD, Dalen JE, McIntyre KM, et al. The electrocardiogram in acute pulmonary embolism. *Prog Cardiovasc Dis.* 1975 Jan-Feb; 17:247-257.

# ECG/VCG DIFFERENTIAL DIAGNOSIS BETWEEN APE AND IMI

|                                         | APE                                                                                                                                                                              | INFERIOR MI(IMI)                                                                                                                                                                                                                                                                                                                                                               |
|-----------------------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Q waves                                 | Eventual significant Q wave in III and aVF.<br>Disappearance of the Q wave in serial tracings                                                                                    | Eventual Q wave $\geq$ 40 ms in DII, DIII and aVF                                                                                                                                                                                                                                                                                                                              |
| Transition zone                         | Characteristic leftward displacement                                                                                                                                             | Localized in intermediate precordial leads V3-V4                                                                                                                                                                                                                                                                                                                               |
| ST segment Injury and reciprocal effect | Slightly elevated in III and aVF.<br>May be elevated in aVR (30%) and accessory Right precordial leads V3R to V6R, similar right ventricular MI.<br>Absence of reciprocal effect | The ECG diagnosis of AMI require at least 1 mm (0.1 mV) of ST segment elevation in the limb leads. The presence of reciprocal changes on the 12 lead ECG may help distinguish. The contour of the ST segment may also be helpful, with a straight or upwardly convex (non-concave) significant ST segment elevation: it resembles monophasic action potential "Pardee Complex" |
| T waves Ischemia                        | Frequent negative T wave in III and less frequently in aVF and eventual in right precordial leads. Rarely pseudo Brugada ECG pattern.                                            | Symmetrical, pointy, wide-based                                                                                                                                                                                                                                                                                                                                                |
| Right ventricular strain on ECG         | Presence of one or more of the following ECG findings in severe cases: complete or incomplete RBBB, S1Q3T3, and negative T wave in V1-V4.                                        | Absent                                                                                                                                                                                                                                                                                                                                                                         |
| VCG on FP                               | When the initial deflection of the QRS loop is directed superiorly the magnitude and duration are lesser than in inferior myocardial infarction                                  | QRS loop of clockwise rotation from right to left with 30 ms above the x line.                                                                                                                                                                                                                                                                                                 |

1. Cheng TO. Right-sided chest-lead abnormalities on EKG in acute pulmonary embolism. *J Natl Med Assoc.* 2003 Sep;95:862.
2. Nunes JP. Pseudo myocardial infarction - A condition in need to be redefined? *Med Hypotheses.* 2009 Oct 23. [Epub ahead of print]
3. Cheng TO. Brugada syndrome vs. pulmonary embolism vs. paradoxical embolism--what are we to believe? *Int J Cardiol.* 2004 Mar;94:119.
4. Marchick MR, Courtney DM, Kabrhel C, et al. 12-Lead ECG Findings of Pulmonary Hypertension Occur More Frequently in Emergency Department Patients With Pulmonary Embolism Than in Patients Without Pulmonary Embolism. *Ann Emerg Med.* 2009 Sep 18. [Epub ahead of print]
5. Todd K, Simpson CS, Redfearn DP, Abdollah H, Baranchuk A. ECG For The Diagnosis Of Pulmonary Embolism When Conventional Imaging Cannot Be Utilized: A Case Report And Review Of The Literature. *Indian Pacing Electrophysiol J.* 2009 Sep 1;9:268-275.
6. Vanni S, Polidori G, Vergara R, et al. Prognostic value of ECG among patients with acute pulmonary embolism and normal blood pressure. *Am J Med.* 2009 Mar;122:257-264.
7. 2005 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care - Part 8: Stabilization of the Patient With Acute Coronary Syndromes" (2005). *Circulation* 112: IV-89-IV-110

# Predictor For Right Ventricular Dysfunction In Acute Pulmonary Embolism: Value of T-wave inversion in Right Precordial Leads

Pulmonary embolism is a common disease associated with high mortality. In-hospital mortality ranges between about 1% and over 30%. Right ventricular dysfunction is an independent predictor of early mortality in normotensive patients with pulmonary embolism. The ECG findings showed T-wave inversion in leads V1 to V3 with a sensitivity of 75% and a specificity of 95%, and a diagnostic accuracy of 80% for the detection of right ventricular dysfunction, with positive and negative predictive values of 95.5% and 73.1%, respectively. These results were better than the biomarkers such as cardiac enzymes or B-type natriuretic peptide (BNP) for the early detection of right ventricular dysfunction.

T-wave inversions persisted throughout the period of RVD, in contrast to a transient SI-QIII-TIII pattern detected during the acute phase only.

T-wave inversion in leads V1 to V3 had the greatest sensitivity and diagnostic accuracy for early detection of right ventricular dysfunction, and normalization of the T-wave inversions was associated with recovery of right ventricular dysfunction in acute pulmonary embolism<sup>1</sup>.

T-wave inversion in leads V1 to V3 had the greatest sensitivity and diagnostic accuracy for identifying Right Ventricular dysfunction in patients with APE<sup>2</sup>.

1. Kim SE, Park DG, Choi HH, et al. The best predictor for right ventricular dysfunction in acute pulmonary embolism: comparison between electrocardiography and biomarkers. *Korean Circ J*. 2009 Sep;39: 378-381.
2. Punukollu G, Gowda RM, Vasavada BC, et al. Role of electrocardiography in identifying right ventricular dysfunction in acute pulmonary embolism. *Am J Cardiol*. 2005 Aug 1; 96: 450-452.

# Main Vectorcardiographic features in APE

1. **Transient CW rotation around the longitudinal axis:** More superior and less rightward orientation of the initial vectors of QRS loop. Occasionally the initial vectors are directed to left.
2. **Frontal Plane:** has CCW rotation more often is observed. The final vector oriented to right. When the initial deflection of the QRS loop is directed superiorly the magnitude and duration are less than in inferior myocardial infarction
3. **Right Sagittal Plane** shows CW rotation of QRS loop.
4. The terminal deflection directed to right, posterior and superiorly, with less magnitude than types C and A right ventricular hypertrophy/enlargement.